Indian Heart Journal xxx (2017) xxx–xxx

Contents lists available at ScienceDirect

Indian Heart Journal

journal homepage: www.elsevier.com/locate/ihj

Original Article

Catheter ablation for electrical storm in Brugada syndrome: Results of substrate based ablation

Abhijeet Shelke[a], Ajit Tachil[b], Daljeet Saggu[c], Masilamani Lawrance Jesuraj[d], Sachin Yalagudri[c], Calambur Narasimhan[c,*]

[a] Department of Cardiology, Krishna Institute of Medical Sciences, Karad, Satara, Maharashtra, India
[b] Department of Cardiology, Lisie Hospital, Kochi, Kerala, India
[c] Department of Arrhythmia and Electrophysiology, CARE Hospital, Hyderabad, India
[d] Department of Interventional Cardiology and Electrophysiology, Kovai Medical Centre and Hospital, Hyderabad, India

ARTICLE INFO

Article history:
Received 1 April 2017
Accepted 29 July 2017
Available online xxx

Keywords:
Brugada syndrome
Electrical storm
Catheter ablation

ABSTRACT

Background: Brugada syndrome (BrS) is known to cause malignant ventricular arrhythmia (VA) and sudden cardiac death (SCD). Patients with implantable cardioverter defibrillator (ICD) may experience recurrent shocks from implantable cardioverter defibrillator (ICD). Recent reports indicate that radiofrequency ablation (RFA) in BrS is feasible, and effective. Catheter ablation of premature ventricular complexes (PVCs) triggering VA and substrate modification of right ventricular outflow tract (RVOT) has been described.

Methods and results: Five patients (4 males, age-23 to 32years) with BrS and electrical storm (ES) despite being on isoprenaline infusion and cilostazol (phosphodiestrase-3 inhibitor)underwent 3 dimensional electroanatomic mapping and RFA. Ventricular fibrillation was easily inducible in two patients. Voltage map of right ventricle was created in sinus rhythm in all patients. Substrate modification of RVOT was performed endocardially in one patient, both endocardial and epicardial in three and only epicardially in one patient. Brugada pattern gradually resolved over one week in all patients post procedure. These patients completed follow up of median 40 months (1.5–70). One patient had inappropriate shock due to atrial fibrillation, one had an episode of VF and appropriate shock 24 months after the RFA. The remaining four patients had no device therapy or VA in device log on follow up.

Conclusion: Abnormal myocardial substrate is observed in RVOT among patients with BrS. Substrate modification in these patients may abolish Brugada pattern on the ECG and prevents spontaneous VAs on long term follow up.

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1. Introduction

Brugada syndrome (BrS) is a genetic disease that accounts for approximately 20% of sudden cardiac death (SCD) in patients with structurally normal hearts.[1] This syndrome is characterized by an ST-segment elevation in right precordial leads (V1–V3) unrelated to ischemia, electrolyte disturbances or obvious structural heart disease. It is accompanied by right bundle branch block (RBBB) like morphology of the QRS.

This condition can cause recurrent malignant ventricular tachycardia (VT), polymorphic VT and ventricular fibrillation (VF).[2] Implantable cardioverter defibrillator (ICD) is the standard of care for symptomatic patients with BrS. Patients with BrS continue to experience frequent ICD shocks, and electrical storm (ES) in them is associated with high mortality and morbidity. Electrical storm is defined as three or more sustained episodes of VT/VF or appropriate shocks from an ICD within 24 h. We report five patients with BrS who underwent successful catheter ablation for frequent ICD shocks and/or ES.

2. Methods

2.1. Patients

Five patients with BrS (4 males; age-23 to 32years) presented with recurrent ICD shocks for VT/VF between August 2010 and January 2017. Three of them had undergone ICD implantation for

* Corresponding author at: Arrhythmia and Electrophysiology Services, CARE Hospital, Road No1, Banjara Hills, Hyderabad, 500 034, India.
E-mail address: calambur@hotmail.com (C. Narasimhan).

http://dx.doi.org/10.1016/j.ihj.2017.07.019
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Please cite this article in press as: A. Shelke, et al., Catheter ablation for electrical storm in Brugada syndrome: Results of substrate based ablation, Indian Heart J (2017), http://dx.doi.org/10.1016/j.ihj.2017.07.019

spontaneous Type I Brugada pattern and resuscitated cardiac arrest, and the other two for arrhythmic syncope with spontaneous type I Brugada pattern.

Electrophysiology study (EPS) and radiofrequency catheter ablation (RFA) was performed under three dimensional (3D) electroanatomic mapping system(EAMS) in all.

2.2. Electrophysiology study and radiofrequency ablation

After an informed consent EPS and RFA was performed under local anesthesia and conscious sedation. Endocardial and epicardial aspects of the right ventricle (RV) including the right ventricular outflow tract (RVOT) were accessed via the transfemoral and percutaneous subxiphoid approaches respectively. A 3D electroanatomical shell of the right ventricular endocardium and epicardium was created using a 3D EAMS (CARTO 3 Navigation System (Biosense Webster Inc., Diamond Bar, CA, USA), or Ensite Velocity Navigation system (NavX; St. Jude Medical Inc., St. Paul, MN, USA)). Abnormal electrograms (EGMs) defined as: fractionated potentials (FP; defined as a continuous multi component EGM with amplitude <1 mV and duration >50 msec), split EGMs (two discrete EGMs separated by an isoelectric interval of >50 msec), isolated late potentials (ILP; defined as EGMs inscribed entirely after the QRS complex) and low voltage EGMs (bipolar voltage <1.5 mv, filtered at 30–300 Hz with a notch filter at 50 Hz) were tagged in sinus rhythm (Fig. 1). Induction of sustained VT or VF was then attempted using the following stimulation protocols: Programmed extrastimulation (PES) was performed in all patients, and up to three extrastimuli were delivered from the RV apex and the RVOT. Programmed extrastimulation using short-long-short (SLS) sequence protocols upto ventricular effective refractory period was performed. When these protocols did not induce VT/VF, a protocol of burst pacing upto 300 ms from the RV apex and the RVOT was used. In case of non-inducibility of VT/VF, these protocols were then repeated from the left ventricle (LV). In case of non-inducibility of sustained ventricular arrhythmia from the RV and LV at baseline, induction was attempted during an infusion of Phenylephrine (0.1–0.5 mg bolus over 1 min followed by an infusion of 0.5–1 μg/kg/min, the end points for dose escalation being achievement of any of the following: 1. Systolic BP $\geq$200 mmHg, 2. increase in mean arterial pressure by $\geq$50 mmHg, and 3. Increase in sinus cycle length by $\geq$25%). Finally an infusion of Propofol(20 mg bolus followed by an infusion of 20–60 mg/ h, was titrated to achieve maximum possible deep conscious sedation without compromising spontaneous respiration). After the initial patient, the mapping strategy was modified to include remapping of the endocardium and epicardium during intravenous infusion of procainamide (10 mg/kg over 30 min), regardless of the findings observed during mapping in the baseline state. This was performed for patients no. 2,3 and 4, but not for patient no 5. Patient 5 had just recovered from a VF storm and was on inotropes and pharmacological provocation was considered unsafe. Stimulation protocols were avoided during infusion of procainamide. Irrigated RFA was performed using an 8F deflectable CELSIUS Thermocool D curve catheter (Biosense Webster, Diamond Bar, CA, USA), or a 7F deflectable Therapy Coolpath medium curve catheter (St. Jude Medical Inc., St. Paul, MN, USA) to deliver 30 W energy at 43 °C catheter tip temperature at an irrigation rate of 2 ml/ min in the epicardium and up to 30 ml/min in the endocardium. Areas of fractionated electrograms and late electrograms(whether observed at baseline or during Procainamide infusion) were ablated during sinus rhythm. Similar stimulation protocols were repeated after completion of ablation. The procedure was defined as successful only if no tachycardia (neither nonsustained/sustained VT/VF or premature ventricular contractions (PVCs)) was induced post ablation.

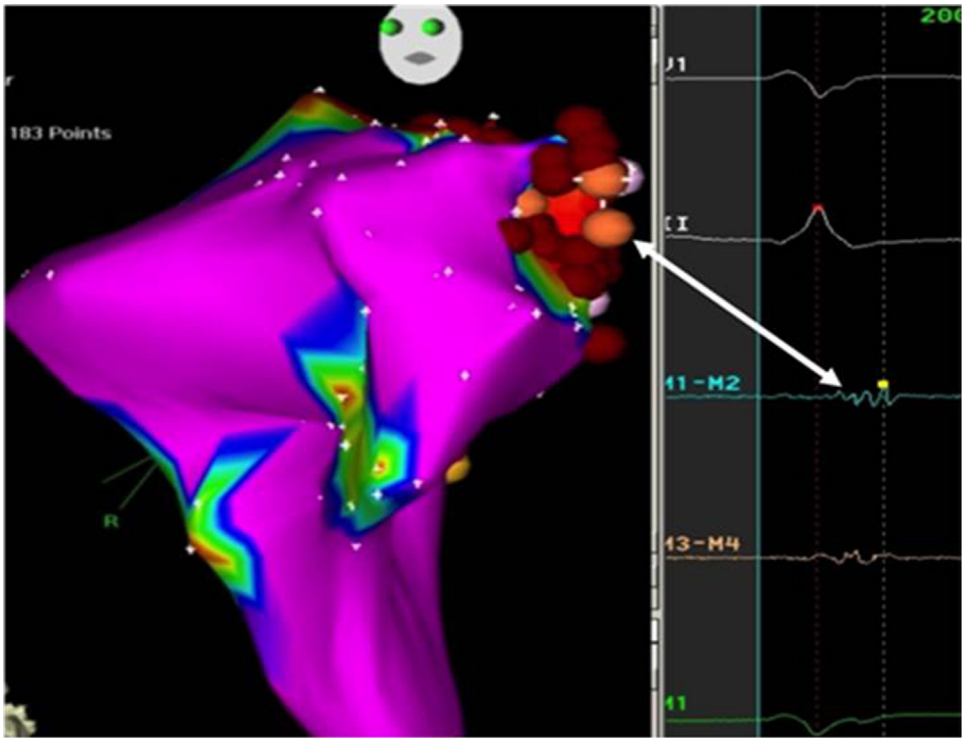

Fig. 1. Voltage guided 3D EAM of RV in LAO view showing: Isolated late potential (white arrow, orange tag) recorded after the QRS from a discrete area of the RVOT septal endocardium in patient no.1; red tag- site of ablation.
3D EAM- three dimensional electroanatomical map; LAO: left anterior oblique view; RVOT: right ventricular outflow tract.

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